THE SYMPTOM-COMPLEX OF A LESION OF THE UPPERMOST PORTION OF THE ANTERIOR SPINAL AND ADJOINING PORTION OF THE VERTEBRAL ARTERIES[1]

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In the June, 1908, number of this journal I described the symptom-complex of occlusion of the posterior inferior cerebellar artery, as it appeared to me from a microscopical study of the material from two cases and the reports of cases in the literature. The symptom-complex is sufficiently characteristic to make a diagnosis easy. In the paper referred to the following occurs:

"Wallenberg sums up Duret's investigations on the supply of the vertebral arteries. Each of these arteries, the left the larger, gives origin about two cm. below their union to the posterior inferior cerebellar artery, and higher to the anterior spinal artery. Branches from the latter artery enter the raphe and nourish the interolivary bundles, posterior longitudinal bundles, hypoglossus nuclei and other nuclei below the floor of the fourth ventricle. The pyramids are nourished by the anterior spinal arteries and frequently by the vertebral arteries. [It is understandable therefore that when the anterior spinal arteries are not occluded the central and anterior parts of the medulla oblongata are not affected.] Branches from the posterior inferior cerebellar or vertebral artery supply the lateral portion of the medulla oblongata."

It has seemed to me desirable to emphasize a clinical picture—the complement of that caused by occlusion of the posterior inferior cerebellar artery—alluded to in my former paper and in my concluding remarks on the discussion of this paper (see this journal, p. 713); especially as I believe I have observed cases that belong to this group. I hope that this brief article may arouse sufficient interest to make the symptom-complex a source of observation and study by others.

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The anterior spinal arteries arise from the vertebrals about a centimeter before the latter unite to form the basilar. The left anterior spinal artery is frequently the larger, and according to Duret the right may be absent. I have found that these arteries sometimes descend several centimeters before uniting, in other instances they unite shortly after their origin to form one vessel. Many of the fibers of the hypoglossus nerves leave the medulla oblongata above the origin of these arteries. Branches of these arteries supply the median portion of the medulla oblongata, especially the lemniscus and the anterior pyramids, although the latter may be nourished also from the vertebral arteries. Duret believed that branches of the anterior spinal arteries supply the hypoglossus fibers, but it seems probable to me that this nerve as well as the vago-accessorius is nourished more from the vertebral arteries. I have seen complete absence of the right vertebral and anterior spinal arteries.

The important fact is that the area of supply of the upper part of the anterior spinal arteries is chiefly the anterior and middle portions of the medulla oblongata. Occlusion of these arteries or hemorrhage from them, including the adjoining part of the vertebral arteries, should therefore give a very definite symptom-group. This may be unilateral or bilateral, depending on the union of the two arteries near their origin or several centimeters below. As these arteries are side by side shortly below their origin, even when they do not at once unite, the symptom-complex is likely to be bilateral; whereas in occlusion of the posterior inferior cerebellar artery it is always indicative of an unilateral lesion.

We should expect to find paralysis of all the limbs, trunk and neck, whereas the reflexes necessary to life would be preserved. The face would not be affected. The tongue might escape, as the hypoglossus nerve has at least a portion of its origin above the origin of the anterior spinal arteries, but it might be involved by implication of the vertebrals. As the lemniscus probably conveys fibers of deep sensation, this form of sensation would probably be disturbed. Henschen believes the lemniscus contains tactile fibers. If this opinion be correct tactile sensation would be affected. If the symptom-complex were unilateral the disturbance of sensation referred to and that of motion would probably be on the same side of the body, as all the fibers concerned with these functions have decussated at the level in question. If the lesion extended lower sensation might be affected on the side opposite to that of the disturbance of motion, or even on both sides of the body. The tendon reflexes are not infrequently lost when the cerebellum or fibers connecting with it are involved, and as in the lesion under consideration the cerebello-olivary fibers would be implicated, it is possible that the tendon reflexes would be lost instead of exaggerated, although this loss might be due to other causes. The vagus and glossopharyngeus should escape, at least in great part. Ataxia probably would not be observed because of the motor paralysis or the escape of important cerebellar fibers.

Occlusion of the vessels in the posterior part of the brain is more likely to occur in syphilitic arterial disease, and transitory quadriplegia might be caused by temporary interference with this vascular supply.

I am well aware that a certain degree of uncertainty is to be expected in the description of a symptom-complex such as this without sufficient clinical and pathological material, and I have delayed the publication of this article many months. The anatomical foundation seems to be reliable, and I am not without some clinical observation. It remains to be seen whether the symptom-complex could be unilateral, as both the motor fibers and the sensory fibers of the lemniscus decussate at about the level under consideration.

I have been able to study a case in the service of Dr. Mills that probably belongs to this type: A woman, forty years of age, previously in good health, attempted to rise from her bed during the night, in April, 1908. She fell to the floor and had the sensation of having no legs, as she expressed it, these limbs feeling numb and dead-like. She was unconscious about two days, and then understood what was said to her, but could not speak for about three weeks. She had incontinence of urine and feces only during the first few days of the attack. She has had a dull aching, tired sensation in all the limbs since the attack. Her speech has been peculiar in that certain words are indistinct and she has difficulty in finding the word she wants at times, and yet she does not appear to be aphasic. She is distinctly weak in all her limbs and equally so in the limbs of the two sides, although the upper are weaker than the lower. The grip is much impaired on both sides, and the fingers are held in partial flexion, but can be straightened by the patient with difficulty, although the wrists remain slightly flexed. The toes are scraped a little on the ground in walking, especially the right, and the lower limbs are slightly spastic; the upper are not so. The tendon reflexes of upper and lower limbs are exaggerated. There are no localized atrophies, and no incoördinate movements. Sensations of touch, pain and temperature are normal everywhere, but the sense of position is distinctly impaired in the toes and fingers, especially on the right side. Babinski's reflex seems to be present at least on the right side.

The patient is intelligent and is positive that she was in good health before the attack, which occurred suddenly and affected both sides of her body simultaneously and equally. The escape of the tongue and face is noteworthy. The important features of the case are spastic paresis of all the limbs with some disturbance of the sense of position.